ment indicates that this proportion approaches the true relative position of the procedure and is in the best interest of the patient.

The mortality rate of 3.5% in the retropubic group is perhaps not a true indication of the results we are now obtaining. Five of these fatalities occurred in the first half of the series. In the last 100 cases there were only two deaths and there have been none in the last 75.

COMPLICATIONS

1. Infection. This is usually an aftermath of urine leakage through the prostatic capsule. It can be avoided by a proper closure of the capsular incision. This has been to us the most difficult part of the operation, and its success is dependent upon: (*a*) working in a comparatively bloodless field; (*b*) making an adequate incision through the capsule according to the size of the gland; (*c*) taking care during the enucleation to avoid excessive tearing of the part to be sutured; (*d*) becoming adept in placing sutures in such a poorly located field.

2. Hæmorrhage. There have been no serious hæmorrhages in this series. Some patients have bled postoperatively somewhat more than others but at no time has this reached alarming proportions. Two cases of secondary hæmorrhage occurred on the 10th postoperative day, but both stopped after inserting a catheter and both patients were discharged shortly after the end of the second week.

3. Suprapubic fistula. Early in the series this was a frequent complication. In the last 150 cases only 3 have occurred which prolonged the hospital stay beyond two weeks.

4. Postoperative stricture. This has not caused any concern since we began removing the V-shaped wedge from the floor of the bladder neck.

5. Incontinence. This complication has not occurred in this series of cases.

6. Osteitis pubis. This occurred once in the first 10 cases following urine leakage with infection. There has been none since.

No assessment of prostatic surgery would be complete without paying tribute to the nursing and intern-resident staff. The after-care of any type of prostatic operation is fraught with a variety of dangers, and the outcome of the operation is frequently decided by the alertness of the nurse, intern or resident on duty. Careful, continuous, and expert attention is essential, and in my opinion, that can only be given by an interested and well-trained hospital in-staff.

CONCLUSION

I have briefly tried to give an impression of our experience with retropubic prostatectomy based on a series of 200 cases in which we developed Millin's technique, varied it slightly as we gained experience, gave the procedure a fair trial, stretched it to the maximum and eventually levelled off to where we now are doing over 40% of our cases by this method. This, we feel, is the proportion of prostatic obstructions which can with safety be handled retropubically and which, when done, gives the patient a smoother convalescence and an excellent final result.

My appreciation is expressed to Dr. F. G. Mack, head of the Department of Urology, Victoria General Hospital, for permission to include his cases in this series for the purpose of a more complete review of the subject.

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ŒSOPHAGEAL VARICES*

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Œsophageal varices present a challenging problem to the surgeon. They constitute the most dangerous complication of portal hypertension; one which is responsible for more than half the deaths in this condition.

It has been known for a long time that varicose veins in the lower œsophagus are secondary to increased pressure in the portal venous system, but the exact mechanism of portal obstruction has been a rather confused subject until recently. The work of Rousselot,[1] Thompson,[2] and Whipple[3] clarifies the picture to a great extent, and it is now apparent that cases of portal bed block fall into two main divisions.

I. *Intrahepatic obstruction* due to obliteration of the portal channels within the liver. This is the commonest form and is exemplified by Laennec's cirrhosis. In this group the liver function is damaged early and this is manifested by bromsulphthalein retention, positive cephalin cholesterol flocculation, impaired hippuric acid synthesis and reversed albumin-globin ratio.

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II. *Extrahepatic obstruction* which has formerly been closely classified as Banti's disease. It is now appreciated that this is not a specific pathological entity but includes a variety of lesions which produce obstruction to the portal blood flow: (1) In the splenic vein, (*a*) trauma; (*b*) thrombosis; (*c*) chronic pancreatitis. (2) In the portal vein, (*a*) cavernomatous change; (*b*) ascending phlebitis from the umbilical veins; (*c*) thrombosis; (*d*) extrinsic pressure.

Effects of portal obstruction.—Hypertension within the portal venous system leads to a characteristic sequence.

1. The raised venous pressure results in congestion of viscera which gives rise to a group of clinical manifestations such as anorexia, splenic enlargement, gastro-intestinal bleeding and anæmia.

2. Collateral channels form between the portal and caval systems in an attempt to compensate for the obstruction. McIndoe[4] quotes H. A. Harris in classifying these into three groups. *Group I.*—At the two situations in the gastro-intestinal tract where the absorbing epithelium comes in contact with the protective epithelium.

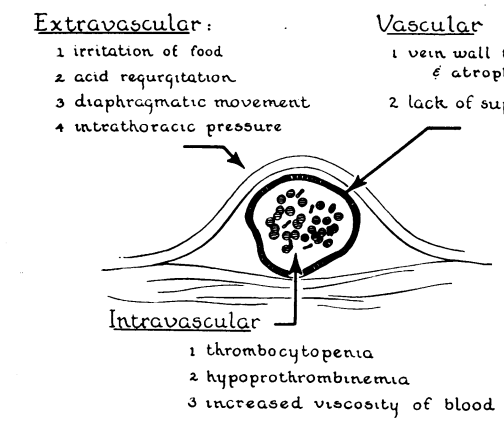

Fig. 1.—Mechanism of bleeding in œsophageal varices.

That is, the lower end of the œsophagus and the muco-cutaneous junction of the rectum. These appear respectively as œsophageal varices and hæmorrhoids. *Group II.*—At the site of an obliterated embryological circulation; the connections of the falciform ligament with the para-umbilical veins. This is manifested by the caput Medusæ. *Group III.*—At all situations within the abdomen where the gastro-intestinal tract and its appendages become retroperitoneal or adherent to the abdominal walls.

The degree of enlargement of such collateral channels varies, but they are seldom adequate to relieve the portal hypertension. In addition, the formation of œsophageal varices constitutes a grave danger to the patient because of the frequency of massive hæmorrhage.

Mechanism of bleeding in œsophageal varices (Fig. 1).—There are many factors which favour the development of varices at the lower end of the œsophagus and in the cardia of the stomach. The connections between the coronary and splenic veins of the portal system and the azygos, intercostal and diaphragmatic veins of the caval system form in the submucosal layer. The vessels lie in a loose areolar tissue which gives little support and in the region of the cardia the veins perforate the submucosa and lie directly beneath the mucous membrane. In portal hypertension reversal of venous flow occurs since no valves are present in the system. The anastomosing collaterals enlarge and protrude into the lumen of the œsophagus. The mechanical factors of constant diaphragmatic movement and changing intrathoracic pressures aggravate this tendency. Tocantins[5] has analyzed the causes of excessive bleeding from œsophageal varices and finds that they are due to abnormalities in all phases of the hæmostatic mechanism.

1. *Vascular defects.*—Due to the mechanical factors mentioned above the vein wall is stretched and atrophied. Rupture may occur from excess pressure within the lumen or traumatic erosion of the vessel wall. 2. *Extravascular defects.*—The mucosa of the lower œsophagus is elevated in longitudinal ridges which are susceptible to trauma with consequent ulceration. Regurgitation of acid gastric juice may also play a part in causing mucosal ulcers. (3) *Intravascular defects.*—Deficiencies in the clotting mechanism exist in many cases of portal hypertension. Long-continued congestion of the spleen leads to increased destruction of platelets and impaired liver function causes hypoprothrombinæmia.

All of these factors predispose to bleeding. Their combination makes the hæmorrhages particularly massive and difficult to control.

The incidence of œsophageal varices and the frequency of fatal hæmorrhage from them is difficult to determine accurately. The veins collapse after death and cannot be visualized except by the injection technique. As a rule this is not done at routine autopsies and consequently, only an approximate figure can be obtained.

River and Wilbur[6] found that in 668 patients coming to the Mayo Clinic with the complaint of hæmatemesis, the bleeding was attributable

to œsophageal varices in 5.1%. In a recent review of 115 cases of œsophageal varices, Higgins[7] reported that 70% entered hospital with hæmatemesis, and of these 84% died of hæmorrhage. At the Royal Victoria Hospital, Montreal, there have been 64 admissions for bleeding œsophageal varices in the past 15 years. Of these patients, 23 have died in hospital, a mortality of 36%. An incomplete follow-up reveals that most of the remainder are dead within 5 years. It is thus evident that the bleeding œsophageal varix presents a serious problem.

Clinical manifestations. — In most cases the first overt sign is a massive hæmorrhage. This follows a fairly characteristic pattern. The patient notices a heavy feeling in the epigastrium and regurgitates bright red blood. The initial hæmatemesis is not vomited but seems to well up into the mouth without retching. This is followed by nausea and vomiting of darker material containing clots. The explanation is that the first blood fills the œsophagus and passes upwards into the mouth and downwards into the stomach. The second phase consists of vomited blood mixed with gastric juice.

Examination of the patient may reveal clinical evidences of cirrhosis of the liver or the Banti syndrome. The diagnosis can only be confirmed by visualization of the varices by œsophagoscopy or barium swallow. Careful x-ray examination with a thin barium paste with the patient in the horizontal position shows the varices bulging into the lumen of the œsophagus producing round grape-like filling defects.

Whipple has emphasized the value of liver function tests in deciding whether portal obstruction is intra- or extra-hepatic. Such tests are also important in the selection of cases for operation. On this basis they can be divided into three groups.

1. Cases in which there is severe liver damage with low albumen, bromsulphthalein retention, positive cephalin-cholesterol flocculation, and in which there is no response to dietary therapy. This is the group of cirrhosis with hepatic decompensation. In such patients the operative mortality is high and little benefit would be expected from relief of the portal hypertension.

2. Cases of cirrhosis with varying degrees of liver damage which show some response to dietary therapy. These patients present a reasonable operative risk and may respond well.

3. Cases of extrahepatic portal obstruction, the Banti syndrome. In these there is usually evidence of marked portal hypertension with little or no impairment of liver function. This constitutes the most favourable group for surgery.

TREATMENT

In acute bleeding from œsophageal varices, the aims of treatment are to arrest the hæmorrhage and replace the blood loss. Transfusions must be given with caution because it has been shown that an increase in the viscosity of the blood tends to precipitate further bleeding. Antacids such as amphogel may be of value in neutralizing the gastric juice which can cause mucosal erosions and interfere with clotting.

If the hæmorrhage cannot be controlled, intraœsophageal tamponnage is indicated. The Miller-Abbott tube makes a satisfactory substitute if the special œsophageal tampon is not available. The tube can be safely left in place for 24 to 48 hours. Food is administered through the lumen and the gastric contents are aspirated periodically to determine when bleeding has stopped. Rowntree[8] and his coworkers have reported several cases in which this method has been used successfully.

When an acute episode has been controlled the question of further treatment becomes urgent. One episode of bleeding is apt to be followed by another and with each successive hæmorrhage the chances of fatality are increased.

The attempts to treat œsophageal varices by surgical means have been directed along three lines, first, the removal or obliteration of the varices; second, the reduction of the portal blood flow; and third, the establishment of more adequate anastomoses between the portal and caval systems. These have all been attempted in human patients with varying degrees of success.

1. *Removal or obliteration of varices.* (*a*) *Injection of varices.*—Crafoord and Frenckner[9] first advocated the injection of sclerosing agents into varices through an œsophagoscope. Moersch[10] has reported 22 cases treated by this means with 12 successful results. The procedure has never been universally accepted. It does

not correct the underlying disease and even if the varices are obliterated they soon recur unless the portal hypertension is relieved. (*b*) *Ligation of the coronary vein.*—It is not difficult to ligate all or most of the veins draining the lower end of the œsophagus but there is no reason to hope that such a procedure will accomplish anything. The varices remain and the high portal pressure is still present. (*c*) *Resection of the lower œsophagus.*—This procedure has recently been recommended by Phemister.[11] It again does not attack the underlying pathological disturbance and offers little chance of permanent cure. Since the basic cause has not been corrected, varices will probably form at the anastomosis. It is still too early to properly evaluate this operation but it appears to be rather radical for such an uncertain basis.

2. *Reduction of the portal flow.*—Splenectomy has been advocated by many writers. The aim of the operation is to reduce the volume of blood flow in the portal system and thus partially relieve the hypertension. Removal of the spleen eliminates 20% of the portal flow but in most cases this is not enough to produce a lasting benefit. In certain patients, however, this operation is curative and the reason for this will be discussed later.

3. *Establishment of porta-caval anastomoses.*—The most rational approach to the problem of œsophageal varices has been directed towards establishing communications between the portal and caval vascular systems. Such communications must be extensive enough to take the dangerous load off the natural anastomoses in the œsophagus. The earliest attempt to create collateral channels was by Talma who recommended omentopexy. This procedure and its modifications have been popular for many years. They have been effective in only a small percentage of cases because although vascular adhesions do form they are not extensive enough to carry any significant volume of blood. Som and Garlock[12] have recently advocated mediastinal packing to create a collateral circulation and have reported good results in two cases. The rationale of this procedure is the same as that of the Talma operation and it would appear to be open to the same criticism.

Von Eck in 1877 first performed a direct anastomosis between the portal vein and inferior vena cava in dogs. He suggested that such a fistula might be of value in treating the portal hypertension of cirrhosis. The operation was not carried out successfully on the human until 1912 when Rosenstein[13] reported a case which was still alive and improved after six months. In the succeeding years, various surgeons attempted the operation but it was not until Whipple[3] and Blakemore[14] published their excellent work in 1946 that the procedure was put on a rational basis. They performed anastomosis between the portal vein and inferior vena cava in some cases, and between the splenic vein and left renal vein in others. They used the non-suture technique with the Blakemore-Lord vitallium tube.[15] The results in a fairly large series have been very encouraging. There is no doubt that such a shunt can relieve portal hypertension if it remains patent. Blakemore[16] and Blalock[17] prefer the Eck fistula type of operation to the spleno-renal because it shunts a greater volume of blood and is thus more efficient in lowering portal pressure.

It is true that the portal stream can be diverted from the cirrhotic liver without producing any immediate harm since most of the blood reaching the sinusoids is carried by the hepatic artery. Thus, the effect of an Eck fistula is not as serious in the cirrhotic as in the normal liver. Nevertheless, any decrease in the blood flow may have grave consequences since hepatic insufficiency will occur much more rapidly with a deficient circulation.

Spleno-renal anastomosis offers several advantages. Removal of the spleen eliminates about 20% of the portal bed and in cases with enlarged, congested spleens an even greater percentage. A spleno-renal anastomosis deviates approximately 40% of the portal flow. In some cases the splenic vein is as large as the portal and a greater shunt may be possible. In any event, a sufficient shunt can be achieved to lower the pressure significantly and to relieve the symptoms of œsophageal varices. In addition, the operation is less hazardous and in the event that the anastomosis cannot be completed no irreparable harm is done to the patient. For these reasons we have adopted spleno-renal anastomosis as the procedure of choice in the treatment of œsophageal varices.

Operative technique.—It is vitally important to give these patients an adequate period of preoperative preparation. A high protein, high carbohydrate, low fat diet with vitamin supple-

ments is maintained for 10 to 14 days. If the prothrombin time is prolonged, vitamin K is given intramuscularly. Methionine may also be of value in improving liver function. Anæmia is universally present and it must be corrected by transfusion and liver extract.

An abdominal approach under continuous spinal anæsthesia has been used in all our cases to date. Linton has recently advocated a thoraco-abdominal incision which appears to give better exposure. When the abdomen is open the venous pressure is measured in branches of the splenic and portal veins with a water manometer. It is most convenient to take readings from the superior mesenteric, the splenic and the coronary vein of the stomach. The normal is 100 to 120 mm. of water. If the pressure is elevated in all vessels, the block must be intrahepatic or in the portal vein itself. The differentiation is made on the basis of liver function tests and the gross appearance of the liver.

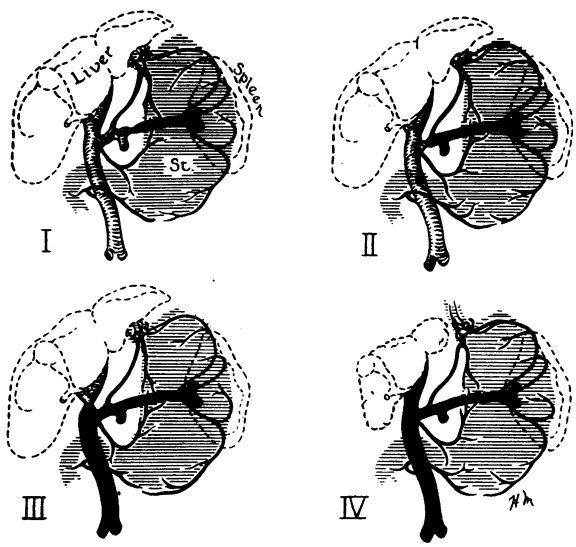

Fig. 2.—Sites of portal obstruction. I. Splenic vein distal to coronary vein: (a) pressure high in splenic and coronary; (b) pressure normal in superior mesenteric; (c) pressure in coronary drops to normal after splenectomy. II. Splenic vein proximal to coronary vein: (a) pressure high in splenic and coronary; (b) pressure normal in superior mesenteric; (c) pressure in coronary remains elevated after splenectomy. III. Portal vein: (a) pressure high in splenic, coronary and superior mesenteric; (b) no change after splenectomy; (c) liver function normal. IV. Intrahepatic—cirrhosis: (a) pressure elevated in splenic, coronary and superior mesenteric; (b) no change after splenectomy; (c) impaired liver function. (I responds to splenectomy alone. II, III and IV require a spleno-renal shunt).

If the pressure is normal in the mesenteric and high in the splenic, the block is in the splenic vein. The site of the obstruction and its relationship to the coronary vein of the stomach determine whether or not a spleno-renal anastomosis is indicated. In a number of cases (10%), the block is distal to the opening of the coronary vein. Thus, when splenectomy has been completed the reverse flow to the œsophageal varices is cut off and the varicosities drain through the unobstructed coronary vein. On the other hand, if the obstruction is proximal to the opening of the coronary vein, splenectomy alone does not remove the backflow of blood and the varices remain distended unless a shunt operation is performed to relieve the portal hypertension (Fig. 2).

An anastomosis must be carried out in all cases of intrahepatic and portal vein obstruction and in those cases of splenic vein obstruction in which the pressure in the coronary vein remains elevated after splenectomy. This group constitutes 90% of the cases of œsophageal varices. In the remaining 10%, splenectomy alone is curative.

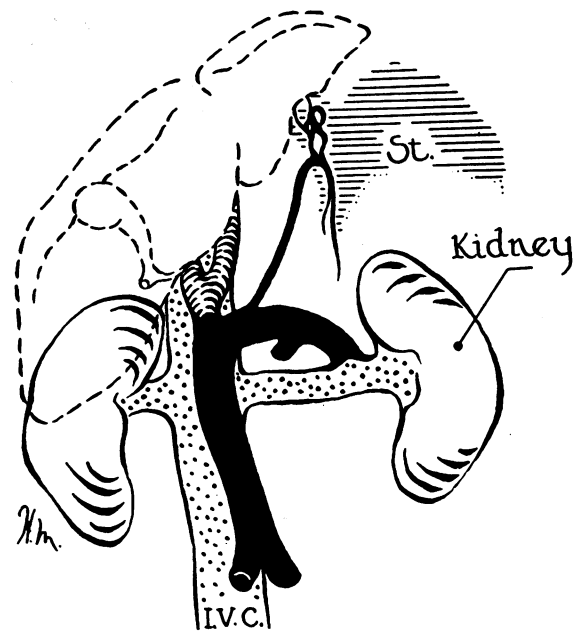

Fig. 3.—The effect of spleno-renal anastomosis on portal vein obstruction.

The spleen is removed, preserving as long a stump of splenic vein as possible. About four inches of the vessel is then mobilized by ligating and dividing the lateral pancreatic branches. The left kidney is exposed and the renal vein and artery are mobilized and occluded with bulldog clamps. It is important to cut off the renal arterial flow during the anastomosis. This can be done for 40 to 60 minutes without causing permanent kidney damage.

If there is no tension an end-to-side anastomosis of the splenic to the left renal vein is done using an everting suture technique. If the splenic vein is short, the left kidney can be removed and an end-to-end spleno-renal anastomosis performed.

The vitallium tube non-suture technique has been abandoned for several reasons. Johns has shown experimentally that there is a lower incidence of thrombosis following a suture anastomosis and we have found this technique to be easier, more exact and simpler to adapt to variations in the size of the vessels. The presence of the stumps of numerous pancreatic branches in the splenic vein makes it difficult to evert a satisfactory cuff over the Blakemore tube and valuable length of vessel is lost by such a manœuvre. In addition, a larger anastomosis can be made by the suture method and no foreign material is present (Fig. 3).

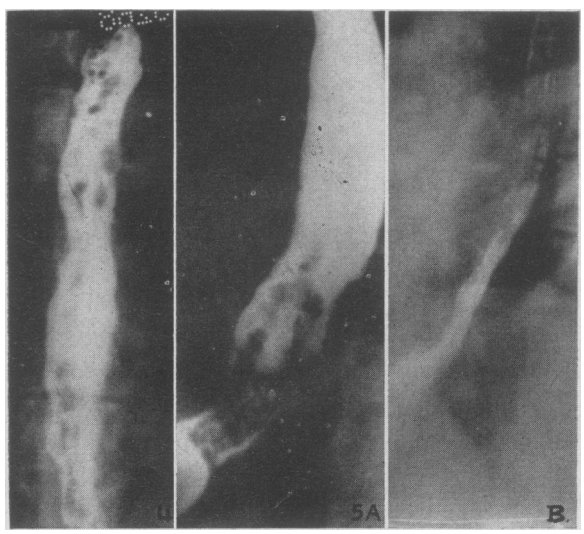

Fig. 4. (Case 1).—Œsophageal varices persisting after three operations which did not correct portal hypertension. **Fig. 5.** (Case 2).—(a) Œsophageal varices before operation; (b) eight months after splenectomy. The varices are reduced in size.

When the completed anastomosis is seen to be patent and functioning, the abdomen is closed without drainage. Postoperative care is the same as for any major abdominal case. Heparin is given for seven days after operation and we have found a dosage of 50 mgm. I.M. every four hours to be satisfactory.

CASE 1 (FIG. 4)

Y.D., male, aged 25. Banti syndrome with œsophageal varices. This patient had 13 admissions to hospital in the past six years for recurrent hæmorrhages from œsophageal varices. Splenectomy was performed in 1942 with no effect. The coronary vein of the stomach was ligated in 1944, and in 1946 all the veins around the lower œsophagus were ligated by a transthoracic approach. Despite these operations he continues to have episodes of hæmatemesis and his œsophageal varices remain unchanged.

Comment.—In this case several attempts have been made to eradicate the œsophageal varices by surgery. None has succeeded. This patient still has portal hypertension and unless this is relieved his symptoms will continue. The patient with œsophageal varices who has already had an unsuccessful splenectomy presents a serious problem. The splenic vein is usually thrombosed and cannot be used. The only alternative is an anastomosis of some comparatively minor tributary of the portal system to a caval vein. This type of procedure is technically difficult and the shunt is often inadequate.

CASE 2 (FIG. 5)

D.M., female, aged 15 years. Banti syndrome with œsophageal varices. This patient had eight admissions to hospital in ten years for œsophageal varices with massive hæmatemesis. In the past year her hæmorrhages had been recurring more frequently. On examination she was thin and pale. The liver and spleen were moderately enlarged. Liver function was normal. Barium swallow revealed large œsophageal varices.

The liver was normal in size, slightly fibrotic. The spleen was moderately enlarged. Mesenteric vein pressure 90 mm. H_2O. Coronary vein pressure 300 mm. H_2O. Splenic vein pressure 350 mm. H_2O. Following splenectomy the coronary vein pressure dropped to 150 mm. H_2O and it was considered that no anastomosis was necessary. The postoperative course was complicated by one episode of hæmatemesis on the 2nd day but was otherwise uneventful.

Since operation the patient has been well with no recurrence of bleeding and has gained 30 pounds in weight. Six months after surgery, x-ray showed a marked decrease in the size of the œsophageal varices.

Comment.—This case represents the type of Banti's syndrome which is benefited by splenectomy. The portal block was in the splenic vein distal to the opening of the coronary vein and removal of the spleen eliminated the source of reverse flow. These cases can only be selected by careful differential pressure readings at operation. The practice of indiscriminate splenectomy for œsophageal varices must be avoided. These patients should not be explored unless the operator is prepared to carry out an anastomosis if it is indicated, as it is in 90% of the cases. Splenectomy does no good in most patients and it eliminates the chance of performing a splenorenal shunt in the future.

CASE 3 (FIG. 6)

L.V., male, aged 40. Banti syndrome with cirrhosis of the liver and œsophageal varices. This patient had his first episode of hæmatemesis at the age of 32. One year prior to his admission to the Royal Victoria Hospital a laparotomy was performed elsewhere and a diagnosis of cirrhosis of the liver was made. He continued

to have episodes of bleeding and was admitted for investigation in February, 1948. Examination revealed an enlarged spleen, the liver edge could not be felt and there was no ascites. Barium swallow showed large varices in the lower third of the œsophagus.

The liver function was impaired. Blood chemistry showed: non-protein nitrogen 27 mgm. %; cholesterol 197 mgm. %; protein 6.4 gm. %; albumin 3.0; globulin 3.4; bilirubin: direct 0.95; indirect 1.8; bromsulphthalein retention 26%; cephalin flocculation 3+; thymol turbidity 7.0.

Operation was performed March 23, 1948. The liver was small, nodular and bile stained, the spleen enlarged and congested. There was venous engorgement in both mesenteric and splenic tributaries. Venous pressure in all was 280 mm. water. The spleen was removed and because the splenic vein was short the left kidney was sacrificed and an end-to-end spleno-renal anastomosis was performed using a continuous everting suture. At the close of operation the anastomosis was patent and blood flowed from the splenic into the left renal vein.

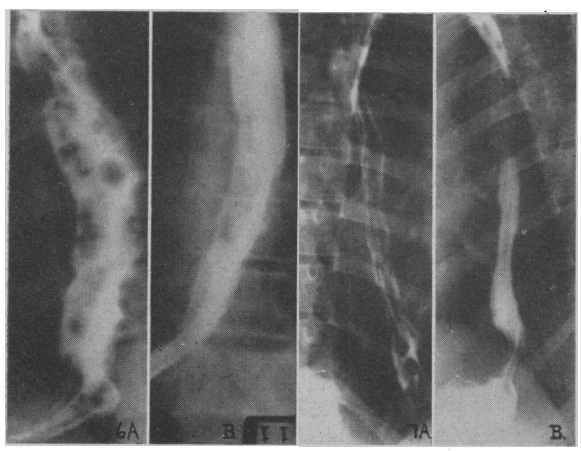

Fig. 6. (Case 3).—(a) Large varices preoperatively; (b) six months after spleno-renal anastomosis. The varices are much smaller and less extensive. **Fig. 7.** (Case 4).—(a) Œsophageal varices preoperatively; (b) one month after spleno-renal anastomosis. There is some reduction in the size of the varices.

Postoperatively the patient was heparinized for five days and made an uneventful recovery. There have been no further hæmorrhages since operation. On the last examination eight months after the anastomosis the œsophageal varices were smaller on x-ray. However, the liver function tests show evidences of more severe impairment.

Comment.—This case represents a satisfactory result in a patient with well marked cirrhotic changes in the liver. The operation has not improved hepatic function but to date the bleeding has not recurred and the prognosis is improved. The varices have decreased considerably in size.

CASE 4 (FIG. 7)

Treated by Drs. D. E. Ross and C. A. Allard at the Children's Memorial Hospital, Montreal.

J.D., female, aged 8 years was admitted to hospital with a history of repeated hæmatemesis for œsophageal varices. Barium swallow confirmed the diagnosis and an end-to-side spleno-renal anastomosis was performed. The postoperative course was uneventful and there has been no recurrence of bleeding in the past eight months.

In an attempt to establish the patency of the shunt, an ingenious method was devised. A catheter was introduced through the inferior vena cava into the left renal vein and by this means the level of the blood sugar in this vein could de determined. It was found that in the post-absorptive phase the blood sugar was consistently higher in the left renal vein than in an arm vein. This indicates that portal blood is being shunted directly into the systemic circulation and the anastomosis is patent.

CASE 5 (FIG. 8)

C.S., a male, aged 59, was first admitted to the Royal Victoria Hospital in 1938 with glycosuria and impaired glucose tolerance. In 1946, the diagnosis of cirrhosis of the liver was established. In 1948 he was readmitted to hospital because of ascites and at that time the barium series revealed large œsophageal varices. Liver function tests showed marked impairment of function with bromsulphthalein retention and a reversed albumin-globulin ratio. He improved somewhat on dietary therapy.

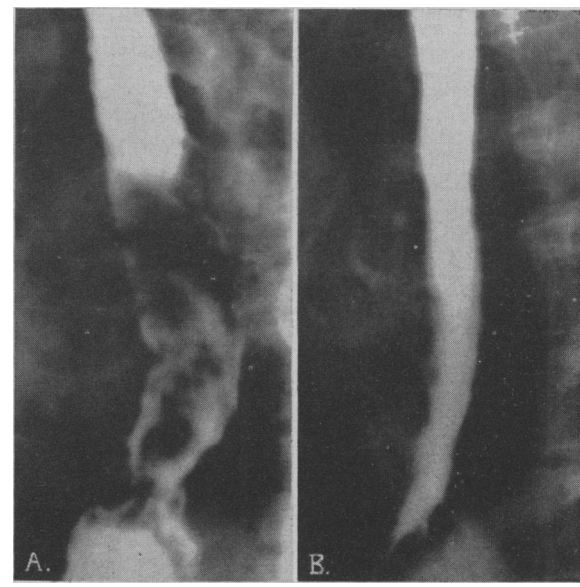

Fig. 8. (Case 5).—(a) Large œsophageal varices before operation; (b) œsophagus two months after spleno-renal anastomosis. The varices have regressed.

Operation was performed on December 6, 1948. The liver was small, hobnailed and bile stained. The portal pressure was 300 mm. of water. An end-to-end spleno-renal anastomosis was performed and at the close of operation the portal pressure had dropped to 180. The patient has made a good recovery; two months after operation his œsophageal varices have regressed and his ascites had decreased considerably.

Comment.—This case responded well to operation despite the impaired liver function. It is recognized that such patients represent poor risks, but in selected cases a shunt procedure is justified as no other form of therapy alleviates the condition.

CONCLUSIONS

Œsophageal varices are a manifestation of portal hypertension and any attempt at surgical treatment should correct the underlying pathological changes.

The most rational approach to the problem is directed towards establishing an adequate col-

lateral anastomosis between the portal and caval venous systems. This is best done by a splenorenal end-to-side anastomosis removing the spleen and preserving the function of the left kidney. Such a procedure diverts approximately 60% of the portal blood flow.

The relief of the portal hypertension may or may not cause the varices to disappear. However, in most cases the bleeding tendency is relieved. Removal or obliteration of the varices would be much more rational as a secondary procedure after the portal pressure has been reduced to a normal level. It is advisable to wait several months before considering such an operation because in many cases the varices slowly decrease in size and are eventually obliterated.

In patients with adequate liver function the operation does not carry an unduly high mortality. This is especially true when the death rate in untreated cases is considered.

The results of operation have been very encouraging to date. A longer follow-up is essential to properly evaluate this approach to the problem.

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One of the remarkable things about medical education is that its teachers are never taught to teach. Nor is this peculiar to medical education. It holds for all professional education, and I gather for most collegiate education as well. It is generally believed that if one has a scholarly grasp of his subject he can *ipso facto* teach it. But this does not necessarily hold. There are methods and techniques which can make teaching more effective, and medical teachers should have some positive training in them.—J. H. Means.

NON-SURGICAL ABDOMINAL PAIN

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THOSE patients who complain of abdominal pain and discomfort constitute a very large group. Although this general discussion concerns those persons whose symptoms arise from conditions primarily treated medically, *e.g.*, the so-called peptic ulcers;* the sphincteric and small bowel dysfunctions; the pre-psychotic states; the obvious and concealed psychoneuroses, and some of the gastritides and colitides, much of it also applies to the surgical patients.

Because the evaluation of any form of therapy is usually made upon the future, as well as the immediate, results, the over-all treatment of these conditions must be considered unsatisfactory. For example, the recurrence rate of duodenal ulcer is 50 to 70% and the incidence of other complications, although less, is often more serious; the various treatments for ulcerative colitis indicate that the prognosis of this condition is not always good; the pre- or actual psychotic, and the psychoneurotic patients are often told there is nothing wrong with them and are thus allowed to progress to the irreversible states of illness, many of which are preventable; the patient with a colonic neurosis often has an appendix removed with no benefit, and sometimes much harm, and the patient with a gastric neurosis is often treated only with medicines. It does not seem, therefore, to be an exaggeration to say that the diagnosis and therapy of some of these symptoms can be improved upon, and that the problem consequently deserves consideration. This paper deals with such a consideration in so far as it concerns a more practical approach.

This approach is based on the thesis that the present therapy often neglects the etiological factors, and is too little concerned with the education of, and an understanding of, the "patient as a person".[1, 2] It also emphasizes the fact that such basic factors should form the foundation of any therapeutic consideration.

* Gastric ulcer is much more of a surgical problem than is duodenal ulcer. It deserves surgical consideration more often than any of the other conditions producing non-surgical abdominal pain. This is because *any gastric ulcer may be a malignant ulcer even if it heals by x-ray and all clinical evidence of its presence disappears.*